RENAL COMPLICATIONS AFTER ANESTHESIA AND OPERATION
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Preliminary Considerations

The concept of complication is so vague that it is necessary to elucidate its meaning before discussing complications limited to an organ, such as the kidney, and within that organ limited to a particular set of circumstances such as anesthesia and operation.

Let us assume that complication is interruption of function. Then, what of those organs with cyclic or intermittent activity? Should a time factor be added? Perhaps interruption of function can be labeled a complication only if it precludes viability. But what of areas of necrosis in an organ leading to permanent interruption of function without loss of life or loss of function for the organ as a whole? Should abnormal function be called a complication? If so, this would imply a complete knowledge of what is normal and what is abnormal.

The latter point may lead to ambiguity. For instance, an acceptable definition of normal kidney function could be that function which eliminates all pharmacologic and metabolic debris which may not be eliminated via the lungs, skin, or alimentary canal. As long as this is accomplished then by definition abnormal function may not be claimed.

The preceding remarks should lead us to deal with complications in a rather sophisticated way; rather than enumerate "complications," we shall review the anatomic and physiologic characteristics of the kidney and the modifications likely to occur during anesthesia and operation. We shall avoid calling such modifications complications because of the ambiguity of this appellation.

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ANATOMY

We shall give particular attention to four anatomical features of the kidneys: the nephron, the vascular pattern, the innervation and the renal capsule.

The Nephron. The nephron (fig. 1) is the functional and anatomical unit of the kidney. It begins at the level of the glomerulus covering its capillary loops with a single layered epithelium which then reflects itself to form the parietal portion of Bowman's capsule. The cavity formed is in direct continuity with the proximal convoluted tubule, which is in continuity with the loop of Henle which is in turn continuous with the distal convoluted tubule. Beyond this point the nephron ceases to exist in the sense that the remaining portion of the system has no effect on the quality or quantity of the finished product, i.e., urine.

For all intents and purposes the structures of the nephron are formed by a single layer of epithelium. This implies that there is no framework to protect, shape or otherwise support the tubule in any of its segments. It follows that tubular patency is maintained by the presence of a relatively incompressible liquid (urine), or by the degree of plasticity of the cells in the tubular wall. The boundaries within which these mechanisms are operative are the physical properties of the renal capsule on one side and the intrinsic tissue pressure and water content of the kidney on the other.

The fluid which collects in the glomerulus is the result of an ultrafiltration process which takes place in the capillary loop of the glomerular tuft. Technically speaking, these loops are a rete mirabile arteriosum and they will be discussed with the vasculature of the kidney. During its journey through the nephrons the large amount of fluid collected in Bowman's capsule, approximately 200 liters a day, is reduced to about 1,600 ml. and its composition is altered by the reabsorption, ex-
cretion or secretion of appropriate substances. It is apparent that the kidney is the seat of intense metabolic activity which must require not only the flow of large volumes of blood per unit of time but also the consumption of a conspicuous amount of oxygen.

The Vasculature. Each kidney receives blood through a single renal artery. Upon entering the hilus of the kidney the artery divides into a number of short interlobar arteries which continue their course in the general direction of the renal surface. As soon as they reach the junction between the medullary and cortical portion of the kidney they bend sharply and run parallel to the outer surface forming arterial arches, hence their name arciform arteries. From these arteries the interlobular arteries emerge in a radial centrifugal direction. From the interlobular arteries arise the afferent vessels which are the feeder vessels to the glomerular circulation. In the glomerulus the afferent vessels divide into a multitude of capillary channels or loops. There are no communications between the various loops which constitute a glomerulus. The glomerular capillary is lined on the outside by a single layer of cells which forms the visceral portion of Bowman’s capsule. In so far as it is known, oxygen is not unloaded or carbon dioxide absorbed at the level of the glomerular capillary. Thus, blood which leaves the glomerulus is still arterial blood. At the distal end the loops converge to form the efferent vessel (fig. 2).

The efferent vessel, shortly after leaving the glomerulus, breaks up into a multiple of true capillaries which supply oxygen to and remove carbon dioxide from the cells which form the renal parenchyma. With the exception of the loop of Henle, the functional portion of all the nephrons is contained in the renal cortex (fig. 1). From the distal ends of the true capillaries venous channels are formed which converge to form a venous system running roughly parallel to the arterial system.

There are four special features to the renal circulation. First, practically all the blood delivered to the kidney must pass through the
glomerular circulation. Second, oxygen required for the metabolic activity of the tubular cell comes from blood which has already circulated through the glomerulus. Third, blood reaches the afferent vessel with a high head of pressure. Fourth, the afferent vessel to the glomerulus is larger than the efferent vessel. Some considerations evolving from the first and second of these features will be discussed later. Features three and four are such that a high pressure gradient may be created easily between the proximal and the distal ends of the capillary loop.

The Nerves. Numerous nerve filaments may be seen in the renal pedicle. The sympathetic innervation originates from the sixth thoracic through the third lumbar segments of the spinal cord, inclusively. The nerve fibers are conveyed to the kidney via the greater, lesser and least splanchnic nerves. Non-myelinated postganglionic fibers may be followed from the celiac ganglion to the arterioles and the glomerular and tubular capillaries. The parasympathetic innervation comes from the vagus.

Stimulation of these nerves produces changes in renal blood flow and urinary output. However, neurogenic activity does not contribute in any major way to the complex mechanism whereby internal homeostasis is maintained.

This is borne out by experimental work in which the kidney was autotransplanted and more recently by reports that a kidney transplanted into a homozygotic twin functions normally even though the nervous continuity never becomes reestablished.

What, then, is the function of the autonomic nervous system in relation to the kidney? Inasmuch as nearly one fourth of the cardiac output may flow through the kidney it should be obvious that some mechanism must be available whereby renal blood flow can be sharply curtailed should a need for blood arise elsewhere. Increased demand for blood flow in other organs undergoing a phase of maximal activity could be one such instance, and acute hemorrhage requiring compensatory vasomotor adjustments in order to maintain flow to other areas may be another instance.

While nerves to the kidneys do not participate significantly in the maintenance of internal homeostasis, in order to produce urine a certain amount of blood must flow through the glomerulus in order to produce a certain amount of glomerular filtrate. In this respect renal nerves may affect urine formation and indirectly internal homeostasis. However, renal blood flow and filtration alone are not sufficient to guarantee the formation of urine

**Fig. 2. Left.** Diagram of the renal circulation. Artery solid black, vein shaded area, renal surface on top (c). IL is the interlobar artery, a branch of the renal artery. ARC is the arciform artery running parallel to the renal surface. ILL is the interlobar artery which runs centrifugally and radially from the arciform artery. (af) is the afferent vessel to the glomerulus (gl) and (ef) is the efferent vessel from the glomerulus (gl). The efferent vessel breaks into a multitude of true capillaries (e) which converge to form venules draining into the interlobar vein which runs parallel to the interlobar artery. Right. Capillary circulation. From the interlobar artery the afferent vessel (af) enters the glomerulus (gl). Shortly after leaving the glomerulus the efferent vessel (ef) breaks into the true capillaries (e). (t) is a tubule although by extension all other parts of the nephron receive blood in the same manner. The capillaries converge to form small veins which then converge to form the interlobar vein (ILV).
because all of the filtrate may be reabsorbed by the tubules. It is also possible that the head of pressure across the glomerulus may not be enough to permit filtration even though flow continues. This always should be borne in mind whenever clearance tests are employed to estimate renal blood flow in the presence of an unstable autonomic nervous system, as in shock, burns, operations, anesthesia, and the like. A wide discrepancy between measured flow and estimated flow (clearance test) in the presence of renal ischemia or intense renal nervous stimulation has been reported repeatedly. It is likely that all conclusions drawn from estimated renal blood flow values should be accepted with reservation.

The Capsule. The renal capsule is formed by strong fibrous tissue with little if any elasticity. This means that it may tolerate great increases in tension without significant changes in linear dimensions.

Other organs which are similarly confined within relatively nonexpandable boundaries are the heart and the brain. We are all aware of the disastrous consequences of acute hemopericardium or acute increases in intracranial pressure. Similarly, we should anticipate that acute intracapsular renal pressure increases are likely to cause serious disturbances in renal physiology. We shall explain later how this increased intracapsular pressure may develop and what the immediate and long range consequences may be. There does exist in the kidney the potential anatomical configuration for the development of a high intracapsular pressure which may lead to collapse of both tubular and vascular components.

**Physiology**

The function of the kidney may be defined as the production of urine for the purpose of eliminating metabolic and pharmacologic debris, including water, in so far as this elimination is compatible with the maintenance of internal humoral homeostasis. For the purpose of the present discussion the type and amount of debris are relatively unimportant. However, what should attract our attention are two basic mechanisms: How is urine formed? How is the formation of urine regulated to changing needs?

**Generalities.** Urine is the end product of three basic processes: filtration, reabsorption, and secretion.

The first step is the production of a glomerular filtrate. This is carried out at the level of the glomerular capillaries and it is brought about by the increase in resistance to blood flow which takes place in that area. Nothing more than filtration, or perhaps ultrafiltration, takes place in the glomerulus. The rate of filtration is regulated by the functional status of the glomerular and capsular (Bowman's) epithelium and by the effective hydrostatic pressure differential across the capillary wall.

All blood flowing through the renal artery must flow through glomeruli before it may supply the parenchyma with oxygen. As the driving head of pressure decreases and provided changes do not take place in the geometry of the vascular system, the amount of filtrate will be reduced. When and if a critical pressure is reached all filtration ceases. At this time urine may not be formed although blood flow through the glomerular capillaries may continue. If the latter were not true, then acute anoxia of the kidney parenchyma would develop promptly. This mechanism may explain why if the arterial pressure is lowered by means of ganglionic blockade relatively long periods of hypotension and oliguria may be tolerated with prompt return of urine formation when the pressure returns toward normal. However, this does not obtain if renal vasoconstriction occurs, either with or without hypotension (see Vasomotion).

Reabsorption and secretion take place in the remaining portions of the nephrons.

Secretion by the tubular epithelium may be construed both as (a) the preparation of a compound which does not exist in the surrounding plasma and its elimination in the urine, or (b) the transfer of a substance from the plasma to the urine involving a diffusion gradient. The production of ammonia is an example of the first type of activity and the elimination of Diodrast exemplifies the second.

In general the reabsorption process follows the same lines albeit in the reverse direction. However, the blood which bathes the tubular cells has a higher osmotic pressure than the protein-free fluid in the tubular lumen. A
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certain amount of reabsorption, particularly of water, therefore takes place independently of specific cellular activity. This occurs in the proximal convoluted tube where 80 to 90 per cent of the filtrate is reabsorbed. This fraction is fixed and independent of all other processes.

The concept of "tubular damage" may be reviewed at this time. Tubular cells may be damaged in any of three ways: first, by presenting to their tubular surface a damaging compound or poison; second, by bringing in contact with them via the blood stream the same or another poison; third, by the interruption of blood flow or oxygen supply leading to cellular anoxia.

With the first two possibilities one would have to postulate the existence of a poisonous substance capable of going through or being filtered at the level of the glomerular capillary without producing damage and yet being able to disrupt the normal anatomy and physiology of the tubular cell. The fact that "lower nephron nephrosis" was accepted in the past as a disease entity indicates acceptance of this view. However, this type of nephrosis is seen after a number of varied injuries and appears to be truly nonspecific.

Although interference with the blood supply produces damage to all segments of the nephron, the severity of the damage and its intensity would be related to the tolerance of the individual structures to hypoxia. Classic studies supporting this view have been published & and confirmed. Tubular cells appear to be highly susceptible to damage from hypoxia.

Regulation of Urine Formation. Basically the kidney is faced with two major tasks, i.e., the reabsorption of water and the maintenance of an adequate acid-base balance.

As stated previously, the regulatory activity exerted by the renal nerves on urinary function is limited to their ability to reduce renal blood flow by vasoconstrictor impulses. This in turn reduces the volume of glomerular filtrate. However, other forms of regulatory mechanism must be postulated. For instance, hormonal systems play a role in such regulation.

Water balance is regulated by the antidiuretic hormone which can be extracted from the posterior lobe of the pituitary.11 The release of this hormone probably is governed by changes in the concentration of colloids in the arterial blood flowing in the area of the internal carotid artery.12

The antidiuretic hormone is of interest also from a purely speculative point of view because of a peculiar set of circumstances. It has been accepted that its action is exerted in the nephron and specifically at the level of the loop of Henle. However, this hormone is likewise present in animals whose kidneys do not possess a loop of Henle or an anatomical counterpart thereof and the hormone still possesses the ability to produce water retention.

The normal sodium balance is maintained through the action of a system of hormones originating in the cortex of the adrenal gland. While water is an important component of the organism and a complex hormonal system to supervise its economy is more than justified, yet it is not quite clear why the sodium ion should have been singled out and a hormonal system devised to keep it in check. Why does sodium appear to be the most important ion? This question should not imply that sodium is more essential than chloride but rather that the organism appears to make a greater effort to mobilize more refined and more powerful tools to control the metabolism of this particular ion.

In the fasting and resting individual the normal metabolic processes are such that acid end-products and water are continuously produced. The volatile acidic debris must be eliminated via the lungs but all of the nonvolatile acidic debris must be eliminated via the kidneys along with a proper amount of water. Any loss of acid will entail the loss of base because the kidney cannot eliminate strong acids as such. It is this loss of fixed base which must be kept at a minimum be-
cause none of it can be produced and it must all come from food intake. It is apparent that the conservation of sodium is of major importance and accounts for nature’s interest in the conservation of this ion.

We may summarize this section and anticipate some of the problems that one will have to face in the management of a patient undergoing anesthesia and operation. For practical purposes, the kidney is the main avenue for water loss and the only filter capable of retaining base and losing acid. The factors which influence kidney function are vascular and hormonal. Both factors respond at all times to the immediate water and electrolyte requirements of the organism to such an extent that renal function cannot be separated from these requirements. Thus we should be concerned not only with the end result of kidney function, i.e., the quantity and quality of urinary output, but also with the prevailing neurohumoral and metabolic situation which will effect, through the release of hormones or the regulation of blood flow, the function of the kidney.

Further Considerations

Anesthesia and operation alone or in combination may upset the normal function of the kidney by interfering with any of three basic mechanisms: (a) changes in renal blood flow, either total flow or flow pattern (including pressure distribution within the kidney), (b) critical alterations in body fluids or electrolytes which may not be immediately compensated for by changes in the functional activity of the kidneys, (c) critical disturbances in the kidney itself which in turn may result in profound alteration of the internal balance of the organism. In relation to the second and third statements, alterations in body fluids or electrolytes secondary to operation and/or anesthesia result in derangements of urinary secretion which are not properly renal “complications” and we should expect that if the primary cause is swiftly eliminated or controlled a return to normal will follow. By and large most of these conditions are preventable. However, if a disturbance involves the kidney itself then it is less likely that it may be brought under control. This is particularly true for primary blood flow disturbances which may lead secondarily to profound alterations in the renal parenchyma. This probably is the most common type of renal “complication.”

VASOMOTION

The renal vasculature responds to a variety of stimuli by readjusting its geometry in such a way that an increase in vascular resistance is observed. Increases in renal vascular resistance have been reported after hemorrhage, after trauma with and without hypotension (fig. 3), after experimentally induced placental abruption, after direct stimulation of the nerves to the kidney in the renal pedicle, after stimulation of the sciatic nerve, and after crushing of the abdominal aorta distal to but in the vicinity of the renal arteries. Many things related to or associated with anesthesia and operation may produce an increase in renal vascular resistance. What this means in terms of kidney viability is not clear. However, an attempt to clarify the subject will be made.

Early investigations in this field were motivated by the high incidence of anuria following abdominal aortic aneurysmectomy. Autopsy material was consistent with a diagnosis of tubular degeneration. The laboratory experiments designed to study this problem yielded kidneys showing changes which could be classified as analogous to those observed in man. Experimentally if the renal vasconstrictor response was prevented by means of mechanical or pharmacological denervation better oxygen extraction occurred and few if any histologic changes were seen. Thus the concept that an anoxic or hypoxic insult is the basis of tubular damage at least under certain conditions came into being.

To clarify the concept a number of experiments were carried out. At first an attempt was made to demonstrate that the oxygen tension in the renal tubule falls after an appropriate stimulus results in marked renal vasoconstriction. This was done by measuring the partial pressure of oxygen in the urine,
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[Graph showing renal and vascular resistance over time]

FIG. 3. Effects of trauma on renal vascular resistance measured as a ratio between pressure and flow. Changes are expressed as percentages of control values. Trauma applied at arrow. The traumatizing technique was 100 blows/10 pounds body weight with a light mallet to the hind limbs.

The delay observed in some instances appears to be due to the state of hydration although no quantitative data on this were obtained.

which reflects peritubular oxygen tension under proper conditions and with proper restrictions. These results indicated that renal vasoconstriction did indeed produce a fall in urinary oxygen tension.

While it could be accepted that this initial hypoxic insult is eventually responsible for the tubular degeneration much remained to be explained. For instance, routine histologic staining failed to reveal changes until the third or fourth day after injury; also these changes were nonspecific. Also, there was no evidence that the normal metabolism of oxygen had been disrupted.

To elucidate this problem, enzymatic staining of the kidney was tried on freshly collected specimens. This technique depends on the staining of precipitates produced by the interaction of certain enzymes and certain highly specific substrates. In this manner the concentration and distribution of certain enzymes can be mapped. Furthermore, with an appropriate selection of enzymes, particular aspects of various metabolic processes may be investigated. By this technique it was possible to prove that within less than two hours abnormal enzyme patterns may be detected and complete disorganization of some oxygen-carrying enzyme systems could be seen early, even though conventional staining yielded normal appearing kidneys.

From the experimental material it was concluded that intense vasoconstriction may produce sufficient anoxia to induce cellular damage and death in the kidney tubules. But where did vasoconstriction occur anatomically? Once this was established one might match the predicted and observed patterns of anoxic tissue death to obtain indirect confirmation of the hypothesis. Studies carried out during direct stimulation of the renal nerves have shown that when vasoconstriction occurs blood is promptly diverted into the systemic circulation. Since it is the afferent vessel which constricts one should expect the pattern of tubular damage to be one of irregular distribution such as to involve most structures in the renal cortex. Experimental work has shown this to be the case and available clinical material supports this hypothesis.

There remain two minor points to be cleared. On occasion the histologic picture leads one to suspect that anuria is the primary etiologic mechanism and that it has been a
consequence of impediment to urinary flow because of the precipitation of materials in the tubular lumen. The other refers to the persistent anuria after re-establishment of an adequate circulating blood volume.

Concerning the first problem, immediately after vasoconstriction occurs, although some filtration continues little if any urine is formed if the appropriate salt and water retention mechanisms are mobilized. In this manner non-reabsorbable material may precipitate in the tubular lumen to result ultimately in obstruction. However, the primary disturbance is vascular. Some results bearing on this subject have appeared in a recent monograph where it is shown that nephrosis from incompatible blood transfusion is made worse if hemorrhage is induced simultaneously. In this case, hemorrhage would act by precipitating vasoconstriction and water reabsorption. This picture is favorably altered by the administration of chlorpromazine which in the opinion of some is an adequate agent to reduce excess autonomic activity. An example of the fact that precipitation of extraneous substances does not necessarily result in tubular blockage may be seen in figure 4.

To explain why the anuric state may persist

![Diagram](image)

**Fig. 4. Left.** Woman, age 43. Presenting symptom, large retroperitoneal suprarenal tumor displacing the liver to the left. Major preoperative complicating factor was that as a consequence of a previous transfusion a high titer of antibody against a minor antigen was present in her blood. After consultation, and in view of the difficulty of securing blood of her own type (B−) antigen free, it was decided to use type O+ blood free of that particular antigen for transfusion during operation.

Anesthesia was induced with thiopental and continued with cyclopropane. After about one hour, while the tumor was being mobilized, profuse bleeding occurred from the liver bed and it became apparent that a large volume of blood was going to be required. In view of this, hemolysis of the patient's own red cells by the α and β antibodies in the donor's blood had to be expected. The combination of hypotension, blood loss, and profound hemolysis was considered to be optimal for the development of postoperative renal shutdown. Accordingly a multilateral attack was devised.

Canglonic blockade was instituted immediately by intravenous infusion of trimetaphan (Arfonad) 0.3 per cent (Arf) in an attempt to prevent renal vasoconstriction. The rate of parenteral fluid therapy was increased using a mixture of equal parts of isotonic glucose and isotonic saline solutions. An intravenous infusion of 5 per cent mannitol (mann) was begun and continued until a satisfactory urinary output was reestablished. Purified serum albumin was administered intravenously to supplement blood transfusion. The operative blood loss, which was completely replaced, was 9,000 ml. In the recovery room it was noticed that the urine had become dark and the mucosa slightly icteric. Right. The highlights of postoperative course are outlined. The bilirubin level of 0.4 mg. per cent was obtained immediately before operation. Notice the large volumes of fluids required to maintain a high urinary output. During the first 24 hours after operation she received about 1,000 ml. of 5 per cent mannitol, the remainder being an isotonic mixture consisting of 2.5 per cent glucose and 0.43 per cent sodium chloride in water. In the following days the glucose/saline mixture represented about 70 per cent of the daily intake. Mannitol 5 per cent was required only occasionally and no more than 150 ml. were administered during any 24 hour period, with the above mentioned exception of the first day. No attempts were made to alkalize the urine at any time. The postoperative course was uneventful in all other respects, and once the bilirubin level began to fall it continued to do so steadily. The patient was discharged on the twelfth postoperative day.
the following explanation is advanced. If the hypoxic episode, caused by inadequate blood flow, has lasted long enough to produce cellular damage one should not expect normal function to resume as soon as flow is re-established. Rather the damaged cell will tend to absorb water and swell. Because of the limitations imposed by the physical properties of the relatively inelastic renal capsule the swelling is limited within the confines of the kidney so as to cause increased intracranial pressure and tubular collapse. Recent clinical studies and experimental work, the latter involving the use of a water plethysmograph, have shown that the administration of a powerful osmotic diuretic not only restores the flow of urine but also prevents cellular swelling and tubular dysfunction.

Thus, disturbances in renal blood flow patterns which may occur during anesthesia and operation are such that they may lead to a true “renal complication” resulting in tubular degeneration. However, this complication is not directly related to the use of any particular anesthetic but rather to disturbances in the renal blood flow distribution pattern. The maintenance of a normal blood flow distribution pattern may be unrelated to systemic arterial pressure and yet closely related to the intrinsic tone of the afferent glomerular vessel.

**PRERENAL ALTERATIONS IN FLUIDS AND ELECTROLYTES**

A partial or complete suppression of urine formation based on alterations in fluid and electrolytes after anesthesia and operation is observed frequently. The literature on this subject is extensive and from it certain notions have originated which in many instances are routinely accepted without question. We wish to cite two examples: (a) “stress, also operation, induces water and sodium retention,” and its corollary, (b) “do not administer saline in the course of anesthesia and operation because the patient is going to retain sodium anyway.” The acceptance of these maxims has been so wholehearted that isotonic saline solution for intravenous use has practically disappeared from many operating room shelves.

It would serve no use to quote references in support of these views. On the contrary we believe that it would be more profitable to inquire whether or not there may be ground for dissension. Arguments either for or against these views should be reviewed carefully.

Operation and trauma induce a certain amount of tissue and cellular breakdown. The disintegration of cells releases large amounts of potassium and protein. For osmotic equilibrium to be re-established and to prevent “explosion” of nearby cells, which would compound the injury and initiate a vicious circle, the following events must occur immediately and simultaneously: (1) removal of the excess potassium ions from the area of cellular damage and dilution with the bulk of body water, (2) redistribution of water into the area to reestablish proper osmotic pressure, (3) diffusion of sodium into that area to re-establish isonionity. So far there is general agreement on the subject of fluid and electrolyte shift associated with trauma and the concept of a “third space” is widely accepted.

As we have seen in a previous section, the economy of sodium is critical because its stores may be replenished only from food intake. Therefore, any sequestration of sodium into a “third space” must call for sharp curtailing of sodium loss. As for the water, the problem is only slightly different in the sense that the normal metabolic processes liberate a certain amount of water which nevertheless is inadequate for the increased immediate need. Thus, promptly after injury, mechanisms are mobilized for the kidneys to retain water also. It is not clear whether this retention is regulated in accordance with the needs or is an inordinate affair, a world unto itself, and in the end a destructive mechanism. Upon the solution of this dilemma will depend the action of the intelligent anesthesiologist who wishes to avoid such “complications” as oliguria and anuria.

The prevailing thought has it that the retention of water and sodium is an independent entity, potentially dangerous to the life and well-being of the patient. Hence, isotonic saline is banished from the operating room, water intake is restricted, and, paradoxically, the oliguric phase after operation too frequently is accepted as an inherent risk. We wish to dissent. It is likely that water and
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**Fig. 5.** Man, age 74. This patient was admitted with a diagnosis of abdominal aortic aneurysm. After blood was typed and crossmatched, adequate routes for intravenous therapy were secured, the patient was taken to the operating room for excision and replacement of the diseased aortic segment.

Upon arrival in the operating room the blood pressure was lowered by the induction of ganglionic blockade with trimethaphan (Arfonad) 0.3 per cent intravenously (Arf) and operation promptly begun. As soon as feasible the abdominal aorta was occluded proximal to the renal arteries in order to secure hemostasis. Shortly thereafter the administration of mannitol 5 per cent was begun although the hourly urinary output could not be determined for lack of a catheter in the bladder. The blood pressure was maintained below 90 mm. of mercury for the duration of aortic occlusion and after release of the aortic clamp it was allowed to rise to approximately normal levels. The blood loss which was estimated to be 5,000 ml. was replaced in the operating room.

The management of fluid replacement deserves some comments. At the time of operation the patient received 3,500 ml. of fluid intravenously of which 800 ml. were mannitol 5 per cent, 1,000 isotonic glucose in water and the remaining 1,700 ml. a mixture of 2.5 per cent glucose and 0.45 per cent sodium chloride. During the following 20 hours he received an additional 2,500 ml. of intravenous fluids of which 1,000 ml. were isotonic glucose and 1,500 ml. the glucose/saline mixture. The urinary output for the first 20 hours immediately after operation was 2,030 ml. and thereafter remained well within normal limits. His postoperative course was uneventful and he was discharged on the eleventh day.

The remarkable fact about this case is the amount of saline solution which had to be administered due to extensive dissection of loose tissue. Tissues thus traumatized are ideal for the establishment of a “third space” whose water and sodium requirements may result in anuria. This compounds the problem created by the intense renal vasoconstrictor response to injury.

sodium are retained only because of these additional extraordinary needs and that therefore both sodium and water should be given until these requirements are matched. Once this has been done, and contingent upon adequate renal function (see next section), water and sodium will be eliminated much as in the usual fashion. The case reports which will be presented indicate that these needs may be great and that they must be met before urinary output can resume.

The truth of this proposition must be sup-
ported at least in three ways if it is to be accepted and if conflicting therapeutic concepts are to be rejected or modified. First, it must be demonstrated that water and sodium retention is self-limited and dictated only by the immediate needs. Second, it should be proved that there is no primary inability of the kidney to eliminate sodium and water if present in adequate amounts. Third, a suitable number of selected clinical cases must serve to prove the point. Figures 4–6 provide this last link of the chain.

We believe that the body reactions to trauma and operation, insofar as they require the retention of additional amounts of water and salt, will result in oliguria and anuria only if these two substances are withheld or administered in amounts too small to satisfy the needs.

In the section immediately preceding, the

![Graph showing urine output and electrolyte levels over time.](Image)

Fig. 6. Man, age 74. This patient was admitted for elective bilateral lumbar sympathectomy because of arterial insufficiency. At the time of admission no areas of necrosis or gangrene were observed in the lower extremities.

Bilateral lumbar sympathectomy was carried out as planned (arrow at bottom left). The operative course was uneventful and it was not marred by hypotension or excessive blood loss. In the following 24 hours it was noted that his urinary output was poor in spite of what was thought to be adequate hydration (4,000 ml of 2.5 per cent glucose and 0.45 per cent sodium chloride solution). The urinary output for the first 24 hours including operation was 235 ml (underlined figure on top). At the beginning of the second day the urinary output disappeared for all practical purposes and it did not respond to an increased rate of fluid administration. After approximately eight hours of anuria, mannitol 5 per cent was administered intravenously and 1,000 ml, given (arrow at top center). Within one hour from the beginning of mannitol administration urinary output was re-established and promptly it exceeded 50 ml/hour. The total urinary output for the day was 1,071 ml.

On the third day the intake and the output were in good balance and from then on the patient made an uneventful recovery. The boxed figures indicate serum electrolyte patterns during thempy.

The course of events in this patient indicates that a period of oliguria followed by a short period of anuria cannot necessarily be equated with tubular degeneration. The facts at hand are not sufficient to establish whether or not kidney function was completely normal on the third, fourth, and fifth days. Nevertheless, his recovery was uneventful and there were no clinical or laboratory signs of renal insufficiency. Also, this case illustrates the fact that after a period of oliguria and anuria conventional fluid therapy may fail to reestablish urinary output whereas mannitol may be quite effective.
prevention of renal damage secondary to disturbances in vasmotion was considered. In the present section, it has been suggested that renal complications due to prerenal factors may be avoided. It remains to be seen what can be done when and if the kidney itself becomes damaged and ceases to function. Can cessation of kidney function be equated with the viability and recovery capacity of the kidney?

**DISTURBANCES WITHIN THE KIDNEY**

A likely evolutionary pattern of renal disturbance after trauma and operation may be outlined as follows. Immediately after injury, intense renal vasoconstriction takes place. The glomerular afferent vessels are primarily involved with the result that glomerular filtration is reduced and the supply of oxygenated blood to distal points is curtailed. For practical purposes the distal points in question are the tubular portions of the nephrons. Along with this vasoconstriction there occurs increased demand for water and sodium reabsorption which effectively reabsorbs most or all of the filtrate. Hence anuria develops immediately after injury, and in some experiments it may be observed within less than three minutes. If corrective measures are delayed for even less than a few hours the kidney will undergo changes such that its function will be lost and death ensues.¹²

The transition from temporary functional renal shutdown to permanent anatomical failure may be explained as follows. Interference with the normal oxygen metabolism may occur as early as within two hours after injury. At this time the renal blood flow is diminished and the tubules are essentially empty. When and if blood flow resumes, the tubular cell will pick up water and swell rather than resume immediate function. Absorption of water by any cell after anoxic injury is known to occur and the pathologic pictures of "tourniquet shock" and "cloudy swelling" are classical examples. The amount of swelling which may be tolerated by the kidney as a whole is limited by the fact that the relatively inelastic renal capsule can stretch very little. Thus, the intrarenal tissue pressure must increase rapidly with consequent obliteration of tubules and capillaries. A vicious circle is thus created leading to continued hypoxia and accelerated degeneration of the tubular cell.

If this hypothesis is accepted it should be clear that oliguria and anuria at least in the early stages are not synonymous with tubular degeneration, and that prompt treatment, at least at this early stage, may be more rewarding than heroic measures later. What type of therapeutic regime should be instituted can only be speculated at the present. However, we shall offer an outline of treatment which has been encouraging and one example of its application will be given.

If the kidney could be decompressed during the acute phase of cellular edema then the course of events leading to tubular degeneration probably could be avoided. Animal experimentation has shown that early decapsulation will prevent increases in renal vascular resistance after trauma or hemorrhage.²² However, bilateral renal decapsulation is not an innocuous procedure and, furthermore, it does not carry a guarantee of success after a protracted period of anuria.

Tentatively, the following hypothesis may be explored. Perfuse the kidney with a substance which may be filtered out by the glomerulus but may not be reabsorbed by the tubular cell. This substance would tend to hold water within the tubular lumen because of its osmotic activity rather than give it up to the cell. Also, on the other side of the cell the osmotic pressure is high because of the increased protein concentration in the plasma as a result of glomerular filtration. In this fashion swelling of the cells might be prevented and the vicious circle interrupted. Preliminary observations²¹,²²,²³ seem to indicate that 5 per cent mannitol is a suitable substance for this purpose and that its trial after proper hydration and with proper precautions is justified (fig. 6).

**SUMMARY**

Anatomical and physiological characteristics of the kidneys have been discussed. The effects of stress on vascular and physiological peculiarities of the kidneys have been reviewed in an attempt to correlate early and late renal complications with stressful situations.
It is suggested that renal vascular constriction in response to trauma along with the increased physiologic need for sodium and water are the basis for both early and late oliguria and anuria following anesthesia and operation. Prophylactic and therapeutic considerations were discussed.

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REFERENCES


ASTHMA Asthmatics may in part become adrenalin fast due to occurrence of respiratory acidosis. After molar lactate was given to 45 adrenalin resistant asthmatics, adrenalin then produced relief. (Blumenthal, J. S., and others: Effect of Changes in Arterial pH on the Action of Adrenalin in Acute Adrenalin-Fast Asthmatics. Dis. Chest. 39: 516 (May) 1961.)

ALDOSTERONISM Hypersecretion of aldosterone causes physiological changes which adversely influence surgical risk. A variety of clinical conditions cause aldosteronism: Conn’s Syndrome (primary aldosteronism) due to adrenal cortical hyperplasia or tumor; essential hypertension; dehydration; nephrosis; cirrhosis; cardiac failure; Cushings syndrome, pregnancy and toxemia, the postoperative state, and emotional stress. The findings are: hypertension; periodic attacks of weakness, muscle soreness, paralysis or tetany; polydipsia and polyuria; decreased sweating. Laboratory findings include: low serum potassium and high serum sodium; metabolic alkalosis; high aldosterone excretion; normal 17-ketosteroid excretion. If the cause of the disease is adrenal hyperfunction, treatment consists of removal of the tumor, or of most of the adrenal tissue. The patient should be prepared for surgery by the administration of potassium and corticotropin, until reversal of the hypopotassemia and alkalosis is accomplished. (Rogers, F. A.: Primary Aldosteronism, A. M. A. Arch. Surg. 82: 683 (May) 1961.)

HEPATO-RENAL SYNDROME A 35-year-old male in perfect health was scheduled for inguinal herniorrhaphy. Induction of anesthesia was achieved by a total dose of 500 mg. of thiopental, 70 mg. of meperidine, 50 mg. of Gallamine and 0.25 mg. of atropine. Maintenance consisted of nitrous oxide, oxygen and halothane. The estimated halothane concentrations using a Boyle vaporizer were two to four per cent. The anesthetic lasted 30 minutes. Neither anoxia nor hypotension was observed during this period. The patient recovered slowly (4 hours) from anesthesia and subsequently had violent agitation followed by profuse vomiting and abdominal distention. Jaundice appeared on the first postoperative day. Urinary output was 700 ml. Increasing jaundice with renal failure was present until death on the third postoperative day. The histological picture of the liver showed globular fatty degeneration of the liver parenchyma without inflammatory reaction. Tubular and glomerular necrosis was present in the kidney. (Vourec'h, G., and others: Hépatonephrite Aligué Mortelle apres Anesthésie Comportant de l’Halothane (Fluothane), Anesth. Analg. (Par.) 17: 466 (Sept.-Dec.) 1960.)